SPECIAL COMMUNICATION

Mechanical and physical factors in lung function during work in dense environments

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Van Liew HD. Mechanical and physical factors in lung function during work in dense environments. Undersea Biomed Res 1983; 10(3):255–264.—The major effects of breathing dense gas during physical exercise in hyperbaric environments can be traced to high airway resistance during inspiration and expiration and especially to an increased tendency for lung airways to become "choked" during expiration. The body's responses to the high resistance include decrease of alveolar ventilation, which leads to CO_2 retention. This hypoventilation is aggravated by poor mixing in the lung because of low diffusivity of gases in the dense environment. Also, there is a tendency for the person to let the end-expired volume of the lung enlarge; this causes a marked increase of work against elastic recoil of pulmonary structures. Because the elastic work occurs during the inspiratory phase of a breath, there is a disproportionate increase of the work of the inspiratory muscles that may lead to fatigue of inspiratory musculature and consequent aggravation of the hypoventilation.

choked airways
diffusive mixing in the lung
dynamic compression of airways
gas density
hyperbaric environments
inspiratory muscle fatigue
maximal flow-volume curves

Physical exercise depends on the linked actions of the pulmonary and cardiovascular systems; it is usually thought that the cardiovascular system is the weaker link in normal environments. In this communication, the case is made for the pulmonary system becoming limiting when density is greater than normal. Changes in the cardiovascular system brought on by high pressure are ignored on the assumption that such changes are weak compared with pulmonary changes, or that they only become manifest at very high pressures. The major pulmonary effects of hyperbaric environments are all more closely related to the density of the gas breathed than to pressure.

There are three important effects of high density on breathing; the person's reaction to these three effects depends on the mechanical actions of the muscular system and the integrative functions of the central nervous system as well as on the mechanics of the lungs, chest, and airways. 1) Inertance of gas flowing through airways increases. Apparently inertance can be expected to change the distribution of ventilation in some situations but not in others (1). There is so little solid information on inertance that it is not discussed further, except to note that inertance effects may sometimes play a role that we are not aware of. 2) Gas-phase diffusivity is inversely proportional to gas density (2, 3). By having air-breathing men at pressures up to 10 ATA take single or multiple breaths of an airlike gas mixture that contained small concentrations of low-diffusivity and high-diffusivity gases, we were able to show that diffusive mixing in the lung is subnormal in hyperbaric environments (4, 5). The result is to decrease the effective alveolar ventilation, as if there were an increased pulmonary dead space. 3) The third effect, greater air flow resistance, increases the effort required to achieve a certain ventilation or reduces the ventilation achieved by a certain effort. In dense gas environments, air flow resistance is higher than normal on both inspiration and expiration (6).

Gas density and airway distensibility interact to make rapid expiratory flows, such as occur in the hyperpnea of exercise, particularly sensitive to density changes. Figure 1 illustrates the "dynamic airway compression" phenomenon that has been studied extensively by pulmonary physiologists and those who deal with pulmonary pathophysiology (7, 8). The open arrows represent intrapleural pressure elevation by muscular force during expiration and the thick black arrows represent the elevation of alveolar pressure above the intrapleural pressure caused by elastic recoil of the lung. Pressure along the airways decreases from high alveolar pressure to atmospheric pressure, so at some point along the airways, the equal pressure point (EPP), the lateral pressure inside equals the intrapleural pressure. The airways may not collapse at the equal pressure point because the tissue may have strength or elastic recoil to resist deformation. At some location downstream, however, the pressure inside the airways is enough

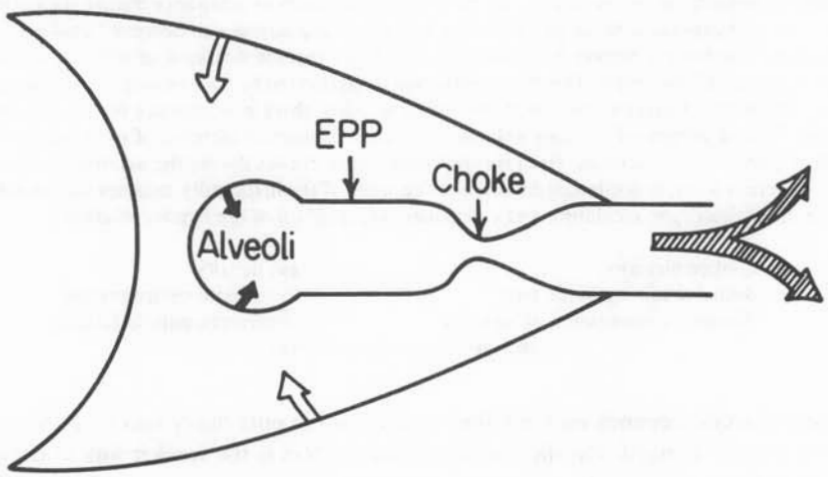

Fig. 1. Schematic diagram of dynamic airway compression phenomenon. During a forced expiration, expiratory muscles generate pressure *(open arrows)* that adds to pressure due to elastic recoil of the lung structure *(thick black arrows)*. At equal pressure point (EPP), pressure inside airways equals pressure outside. At some point mouthward of EPP, pressure outside exceeds pressure inside by enough that "flow limiting segments" or "choke points" develop. [Redrawn from West (7).]

less than the intrapleural pressure outside that so-called choke points develop. When this occurs, flows through the airways are limited and effort independent, meaning that additional pressure exerted by respiratory muscles will not increase the flow. Additional pressure will change both alveolar and intrapleural pressure so that the transmural pressure at the location of the choke point is not changed at all.

The square root relation. The importance of dynamic airway compression to hyperbaric exposures is that, all else being equal, the flow through a choked airway is expected to be proportional to the reciprocal of the square root of ρ, the gas density (9–13). The beautiful simplicity of what can be called the *square root relation* gives a valuable point of reference for interpretation of observations made in hyperbaric exposures.

Figure 2 is a theoretical scheme to illustrate how maximal oxygen uptake ($\dot{V}O_{2max}$) and maximal voluntary ventilation (MVV) can be expected to be related to gas density. The solid circle indicates the minute ventilation of a person who is exercising at the rate at which oxygen uptake is maximal. The curve, drawn from the square root relation, represents MVV as a function of gas density (14), assuming that the MVV (or perhaps better, the maximal sustainable ventilation) is limited by the dynamic airway compression phenomenon. The curve is consonant with many observations (13–16) that maximal ventilation falls rapidly at densities only slightly above normal (at left in diagram) but is not very sensitive to changes of density when density is high (at right in diagram). The solid circle lies below the MVV curve, consonant with the idea that cardiovascular factors, not ventilatory ones, usually limit the $\dot{V}O_{2max}$ in normal environments. The vertical arrow represents compensations in oxygen extraction from the lung, cardiac output, oxygen extraction from the blood, and toleration of moderate hypoxia and of moderately elevated carbon dioxide that may allow the person to get by on less than optimal ventilation but still have the same $\dot{V}O_{2max}$.

The open circle in Fig. 2 shows the minimal ventilation a person could have, despite all kinds of compensations, and still have the same $\dot{V}O_{2max}$. As gas density increases above normal there is a shaded area in which the person can have various ventilations and still maintain the same $\dot{V}O_{2max}$. However, when the density is so high that the person reaches his MVV curve, his performance is limited by ventilation and his $\dot{V}O_{2max}$ must fall in proportion to the way his maximal ventilation falls; that is, according to the square root relation. Data reported by Linnarsson and Fagraeus (17) suggest that air-breathing men exercising at 6 ATA are near the lower right corner of the shaded area of the figure; the subjects' $\dot{V}O_{2max}$ values were actually about 3% higher than at 1 ATA, but expired ventilation during the maximal test was only 80 liters/min instead of the 1-ATA value of 150, and end-tidal P_{CO_2} was elevated.

It is possible to test the square root relation with data from the literature. The uppermost curve in Fig. 3 shows a maximal expiratory flow-volume (MEFV) curve: mean data for 8 men studied at 1 ATA by Hesser et al. (18). During the downslope of such MEFV curves the airways are in the effort-independent state because of formation of choke points. It is clear from the curve that when airways are smaller because the lung is smaller, maximal flow is less. The dashed curve in Fig. 3 shows the prediction for air breathing at 6 ATA calculated from the square root relation by multiplying each flow of the 1-ATA curve by a factor derived from the square root relation. The lower solid curve shows data measured at 6 ATA in the same subjects. After the first 20% of the vital capacity had been exhaled, the square root relation provided an excellent prediction, but higher than 80% of the vital capacity on the volume scale the data flows were far above the predicted. There are probably two parts to the explanation of this discrepancy. First, the flow resistance at 1 ATA is so low that about 20% of the vital capacity can be expired before the dynamic compression phenomenon starts. Second, at 1 ATA the muscles of expiration shorten so rapidly at first that they cannot develop much

pressure on the gas (19). This high flow at high lung volumes that occurs in hyperbaric exposures can be considered an advantage; a person can have higher peak flows than would be expected from 1-ATA measurements.

In addition to obtaining the MEFV data shown in Fig. 3, Hesser et al. (18) had their subjects do heavy exercise. At 1 ATA the exercising men had peak expiratory flow rates of about 6 liters/s; reference to Fig. 3 shows that at that flow, the men's expirations were not seriously limited by dynamic airway compression. The figure also shows, however, that flow at 6 ATA could not exceed 5 liters/s and over most of the volume range flow would have to be 4 or 3 liters/s. The men were forced by the hyperbaric environment to decrease their expiratory flow rates, so their total ventilation decreased. Among the adaptations and compensations observed in the study (18) were low respiratory frequency, conservation of energy by generating intrapleural pressures no greater than needed to cause flow limitation, and, in particular, increase of end-expired lung volume, or increase of the functional residual capacity (FRC), so that the breaths were positioned in the highest flow regions to the left of the middle of the 6-ATA data curve of Fig. 3. Enlargement of FRC during exercise in dry hyperbaric exposures has been observed by several investigators (13, 18, 20, 21), but in immersed subjects the pressure of water on the thorax may prevent its occurrence (22).

Although distension of airways in enlarged lungs decreases airflow resistance and increases diffusive conductance, there are at least two disadvantages to a large FRC. First, pressure-

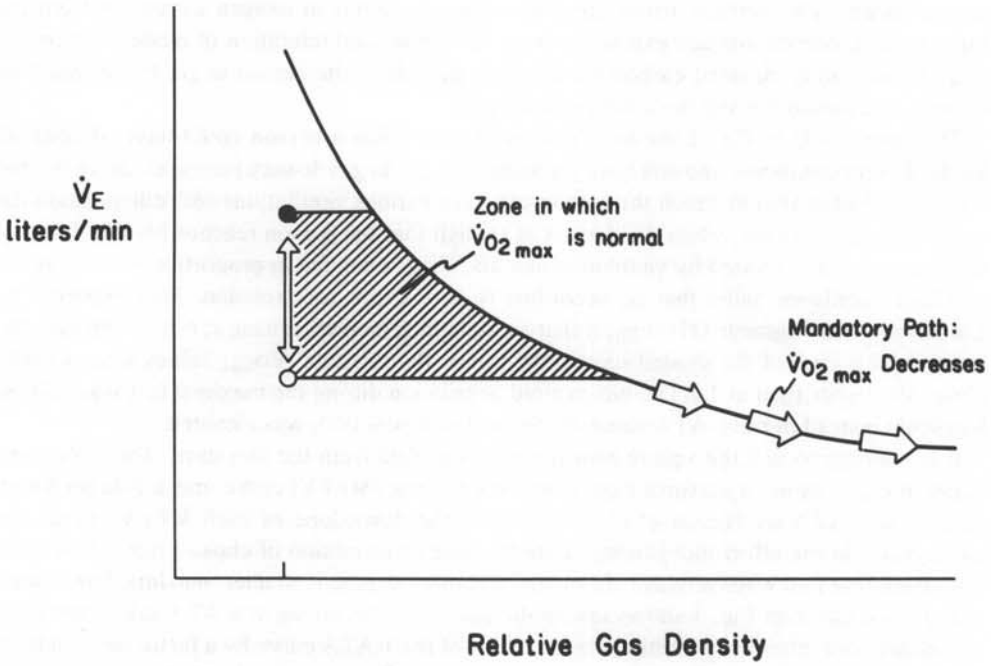

Fig. 2. Schematic diagram for prediction of relation between maximal voluntary ventilation (MVV) and maximal O_2 consumption ($\dot{V}_{O_{2max}}$). *Curve,* MVV drawn according to square root relation, showing fall of MVV as density (relative to air density) increases. *Solid circle,* ventilation normally seen at 1 ATA when O_2 consumption is maximal. *Open circle,* least ventilation at 1 ATA that can be associated with a normal $\dot{V}_{O_{2max}}$. *Shaded area,* zone in hyperbaric environments in which $\dot{V}_{O_{2max}}$ can be the same as at 1 ATA. *Open arrows,* path that ventilation must take at high gas densities; $\dot{V}_{O_{2max}}$ is limited by ventilation and is reduced below normal.

volume characteristics of the lung-chest complex dictate that there is a great increase of work against elastic recoil forces if a person does not return to the resting volume of the lung-chest complex at the end of the breath. The increased effort is expended during inspiration. As illustrated in Fig. 4, elastic forces can be several times larger than normal, leading to a great increase of the work load and consequent increased needs for oxygen and metabolites of the inspiratory muscles. Paradoxically, the inspiratory musculature tends to suffer for the choking off of expiratory flow.

In addition, an enlarged lung puts the inspiratory muscles in a shortened, inefficient position; especially, the diaphragm may be flattened rather than domed, so that diaphragmatic contraction is inefficient for causing inspiration. Because expiratory flow is limited, it would be advantageous to have inspiratory flow very rapid and inspiratory duration short so that more time could be used in expiratory flow. However, inspiratory time has not usually been found to decrease in hyperbaric exposures (18, 21), probably a consequence of the disadvantageous position and the tendency of inspiratory muscles to fatigue.

Diffusive mixing. When subjects inspire a breathing mixture that contains low concentrations of helium and sulfur hexafluoride at normal pressures, the patterns of the two tracer gases during expiration are nearly the same when concentrations fall from the inspired level to the alveolar plateau, but in a hyperbaric environment the expired pattern for SF_6 was

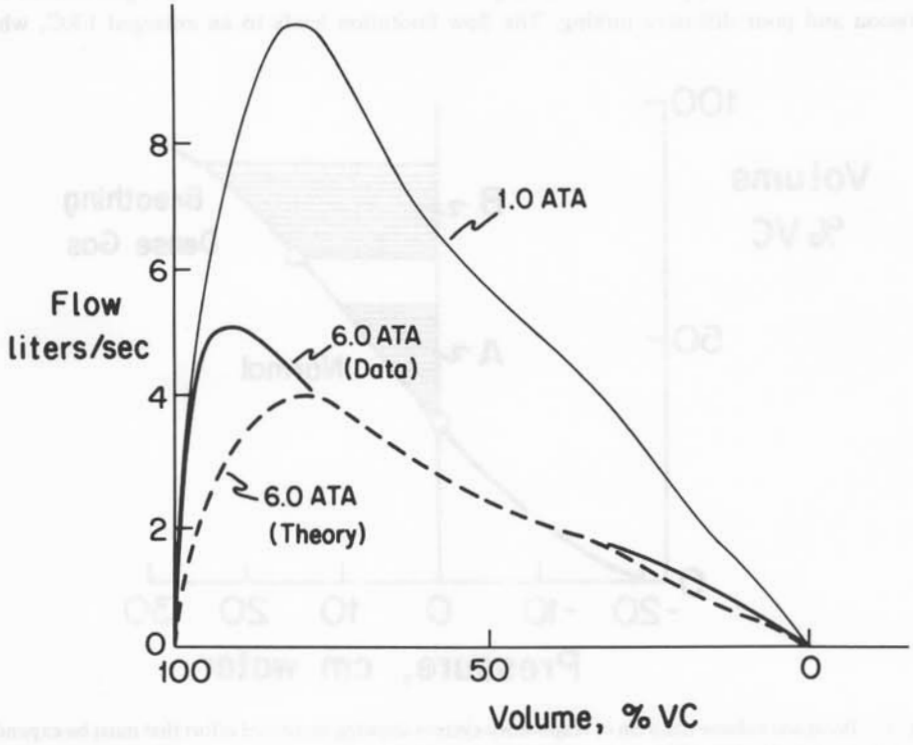

Fig. 3. Maximal expiratory flow-volume (MEFV) curves. *Upper curve,* MEFV for men at normal pressure. *Dashed curve,* theoretical MEFV curve at 6 ATA breathing air predicted from the 1-ATA curve and the square root relation. *Lower solid curve,* data for men at 6 ATA. [Curves from data of Hesser et al. (18).]

changed markedly (Fig. 5). Since the concentration of the poorly diffusible SF_6 is high relative to He throughout almost all of the expiration, it is clear that more He than SF_6 was exchanged between inspired gas and residual gas in the lung (4, 5).

Diffusive hindrance to gas mixing in dense gas environments followed the simple relation

$$A/A_I = B - (0.0010/D) \quad (1)$$

where A is amount of a particular gas exchanged between the inspirate and FRC in one breath, A_I is the amount of the gas inspired, B is the hypothetical optimal fraction of inspirate that would be retained if diffusivity were infinite, and D is molecular diffusivity (5); the appropriate value of D for a particular gas mixture and a particular environmental density can be calculated by the Wilke equation (see Ref. 3). Since Eq. *1* describes a straight line, any decrease of diffusivity will be expected to cause hindrance to diffusion, with no threshhold. To cite an illustrative example, at 10 ATA of air, the diffusivity of O_2 will be 10% of its normal value of 0.25 cm^2/s. The formula predicts that for a breath in which inspired O_2 would be 55% retained at 1 ATA, the value would fall to 51% at 10 ATA. This is not a large change of amount for a 10-fold change in diffusivity. This sort of decrease of effective mixing can be considered an increase of dead space, so low diffusivity could be compensatetd by a simple increase of ventilation. However, because of the effect of dense gas on air flows, the low diffusivity effect adds "insult to the injury" of hypoventilation brought on by high air flow resistance.

Interactions. The upper left of the Fig. 6 scheme indicates that dense gas causes flow limitation and poor diffusive mixing. The flow limitation leads to an enlarged FRC, which

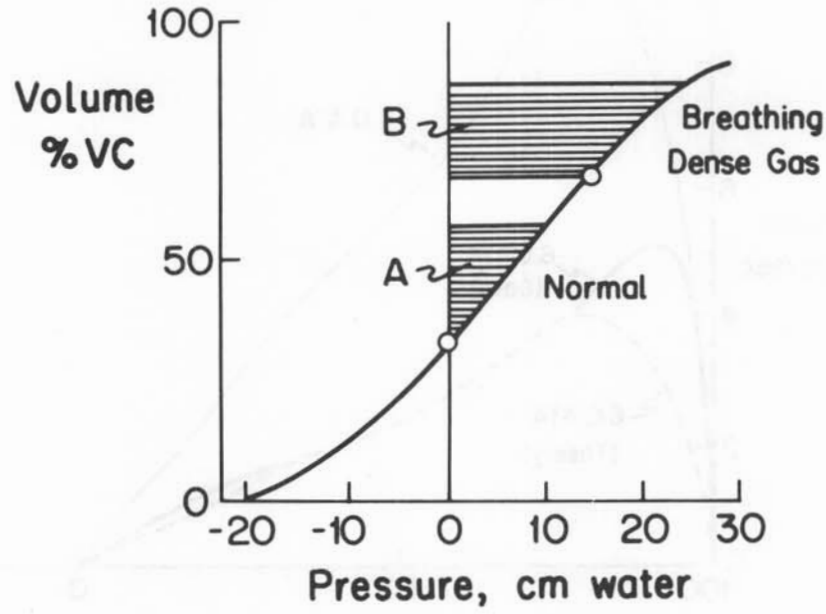

Fig. 4. Pressure-volume diagram of respiratory system showing increased effort that must be expended against elastic forces when FRC is increased. *Lower circle,* resting volume of system. *Hatched region A,* elastic recoil forces for an inspiration that originates from resting volume. *Upper Circle,* enlarged end-expired volume that may be adopted by a diver when he encounters strong resistance to expiratory flow. In such a case the diver saves effort against flow resistance and saves time by obtaining high flows (Fig. 3), but region *B* shows that effort against elastic recoil may be increased by a factor of four or more.

increases effort required by inspiratory muscles, and may lead to inspiratory muscle fatigue. The flow limitation and fatigue tend to cause low alveolar ventilation that is made even lower by the virtual increase in dead space caused by low diffusivity. A reduced alveolar ventilation, as indicated by CO_2 retention, is a common finding in hyperbaric environments, even at rest (14, 16).

The limiting factor that causes men to collapse or to stop work can be expected to be inability to eliminate CO_2, acidosis, and CO_2 narcosis unless something more drastic happens first. One of these drastic possibilities, hypoxia brought on by hypoventilation, is usually not a problem because the breathing medium usually has a high O_2 concentration. Another drastic possibility is shown at the right in Fig. 6; many investigators have reported that unendurable dyspnea caused men to stop working in high pressure environments (13, 16–18, 20, 22). It is not clear what causes the dyspnea, but surely perception of inspiratory difficulty or fatigue of inspiratory musculature would be prime suspects.

The central nervous system (CNS) may play a role in the enlarged FRC and the hypoventilation. Presumably the CNS integration of many kinds of information from sensory structures in the body results in decisions to let the FRC enlarge and to let the CO_2 of body fluids rise in order to conserve energy even when the system is not taxed to its ultimate limit.

Figure 7 shows data from 3 published experiments in which men did physical exercise at or near their maximal capacity, in widely different environments (15, 16, 18). The band was drawn according to the square root relation and was made wide enough to encompass all the data points at densities greater than normal. An interpretation of this figure is that the ability to do steady-state physical work in hyperbaric environments decreases in proportion to the square root of the gas density, as expected if the primary problem is imposed by gas flow through choked airways. Various secondary effects and compensations probably alter a subject's position within the overall downward trend of the band. Finally, since the subjects represented

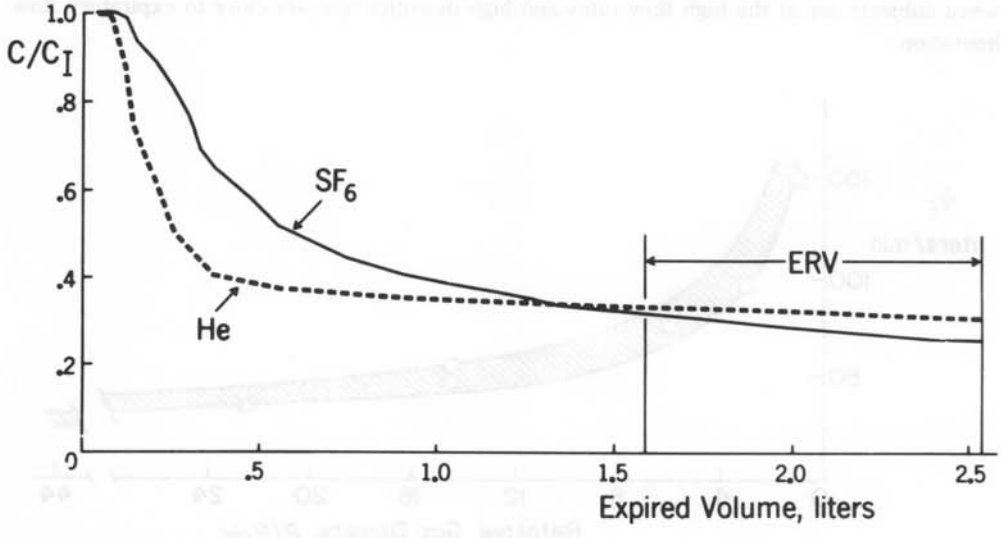

Fig. 5. Expired concentration as function of expired volume after inhalation of 1.6 liters of mixture containing low concentrations of He and SF_6 for young man at rest at 9.5 ATA (5). Concentration is normalized by dividing by inspired concentration, C_I. Helium can easily be considered divided into unmixed dead space gas (at *left*); and well-mixed alveolar gas (at *right*); SF_6 shows effects of poor diffusive mixing whereby it tends to be expired rather than retained in the lung.

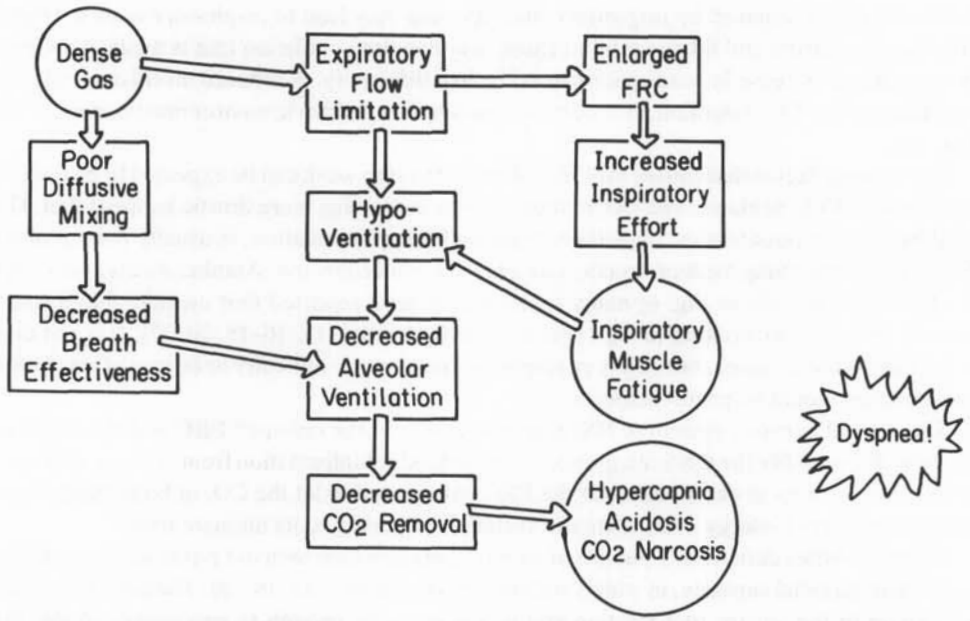

Fig. 6. Schematic diagram of ramifications of poor diffusive mixing and expiratory flow limitation that occur during exercise in hyperbaric environments. Note the paradox that because a major effect of breathing dense gas is to cause expiratory flow limitation, the inspiratory muscles are apt to become fatigued.

by circles in the figure experienced dyspnea (16) whereas those represented by triangles apparently did not (15), it may be that dyspnea is a secondary phenomenon that only occurs when subjects are at the high flow rates and high densities that are close to expiratory flow limitation.

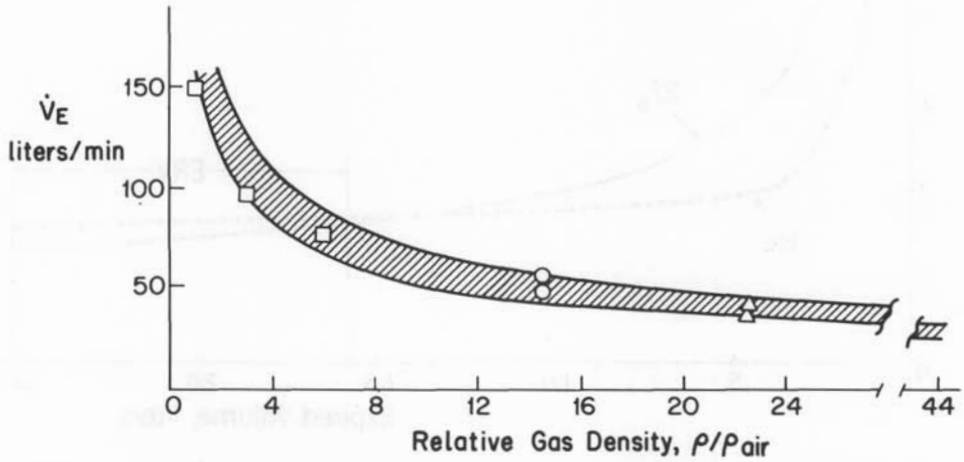

Fig. 7. Ventilation vs. gas density according to square root relation. Width of band was drawn to account for variability and compensations, but overall generalization is that ventilation can be expected to fall approximately as the square root of gas density, and consequently oxygen consumption and work capacity must fall as well. Breathing gases: *squares*, compressed air (18); *circles*, He-O_2-N_2 mixture (16); *triangles*, Ne-O_2-He mixture (15).

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Van Liew HD. Facteurs mécaniques et physiques de la fonction pulmonaire durant le travail en milieux denses. Undersea Biomed Res 1983; 10(3):255–264.—Les effets principaux produits par la respiration de gaz denses au cours de l'exercice physique en milieux hyperbares peuvent être reliés à la grande résistance respiratoire lors de l'inspiration et de l'expiration, et spécialement à la forte tendance à "l'étranglement" des voies respiratoires pulmonaires durant l'expiration. Les réponses corporelles à la haute résistance incluent une diminution de la ventilation alvéolaire, laquelle conduit à la rétention de CO_2. Cette hypoventilation est aggravée par le pauvre mélange dans les poumons à cause de la faible diffusibilité des gaz en milieux denses. De plus, le sujet a la tendance à laisser croître le volume pulmonaire en fin d'expiration; ceci produit une élévation marquée du travail contre le recul élastique des structures pulmonaires. Vu que le travail élastique survient durant la phase inspiratoire de la respiration, il se produit une augmentation disproportionnée du travail des muscles inspiratoires. Ce phénomène peut conduire à la fatigue de la musculature responsable de l'inspiration et à l'aggravation conséquente de l'hypoventilation.

étranglement des voies respiratoires
densité des gaz
mélange diffusif dans les poumons
milieux hyperbares
compression dynamique des voies respiratoires
fatigue des muscles inspiratoires
courbes de débits-volume maximaux

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